Kardiologia Polska 2016; 74, 6: 598; DOI: 10.5603/KP.2016.0091 ISSN 0022–9032

STUDIUM PRZYPADKU / *CLINICAL VIGNETTE*

Ictal asystole: an opportunity for pacing

Asystolia w przebiegu padaczki — czy wskazanie do stymulacji?

Rafael S. Acunzo[1], Rita B. Tepper[1], Nathaniel Moulson[2], Piotr Kukla[3], Adrian Baranchuk[2]

[1]Division of Cardiology, Hospital José M. Ramos Mejía, Buenos Aires, Argentina
[2]Division of Cardiology, Kingston General Hospital, Queen`s University, Kingston, Ontario, Canada
[3]Department of Cardiology and Internal Medicine, Specialist Hospital, Gorlice, Poland

We present a case of a 48-year-old male patient who was assessed and treated for temporal lobe epilepsy. The patient had no further significant past medical history. Nuclear magnetic resonance imaging of his brain was performed as work-up for the aetiology of his epilepsy and revealed a right medial frontal lobe area suggestive of a haemangioma and a right parietal lobe hypodense area suggestive of a vascular lesion potentially due to embolism. A 24-h Holter monitor was then ordered, to rule out asymptomatic episodes of atrial fibrillation as a potential cause of these lesions. During the Holter monitoring, the patient had a witnessed tonic-clonic seizure. Twenty seconds before the episode, the Holter showed sinus rhythm at 60 bpm. His heart rate progressively increased to 120 bpm, and muscle artefacts were observed (Fig. 1A). This was considered the onset of his clinical seizure. His heart rate increased to 150 bpm ictally (Fig. 1B–D), and subsequently decreased to 130 bpm once the seizure activity ceased (Fig. 1E). The episode lasted 66 s (Fig. 1B–E). Following this, his heart rate decreased and a junctional rhythm with junctional premature contractions was observed (Fig. 1F). His heart rate then decreased to 30 bpm (Fig. 2A), followed by the development of sinus pauses (Fig. 2B) and an episode of asystole, lasting 11 s (Fig. 2C). This was interrupted by a junctional rhythm at 22 bpm (Fig. 2D) and sinus rhythm was then re-established after a 3-min period (Fig. 2E). In this particular case, the neurology team felt that medication could be optimised before implanting a pacemaker. In a 12-month follow-up the patient was symptom free. No pacemaker was implanted at this time. Ictal asystole is a potential mechanism of sudden death in epilepsy, and refractory ictal asystole has been proposed as a potential indication for pacemaker insertion in select patients. The Holter tracings in our patient clearly illustrate the previously described cardiac electrophysiological phenomenon of ictal tachycardia, followed by a profound ictal bradycardia culminating in asystole. The mechanism underlying these alterations is felt to be secondary to an imbalance in the autonomic nervous system caused by seizure activity. In patients with sudden death in epilepsy a previously unrecognised cardiorespiratory pattern has been shown lately, including an initial elevation of heart and respiratory rates, followed by central apnoea and profound bradycardia, typically culminating in asystole. It is proposed that ictal asystole is triggered by potent vagal stimulation via brainstem autonomic reflex centres. Pacemaker insertion may be indicated in patients with epilepsy that is difficult to control, despite optimal medical management to reduce the potential increased risk of mortality associated with this phenomenon.

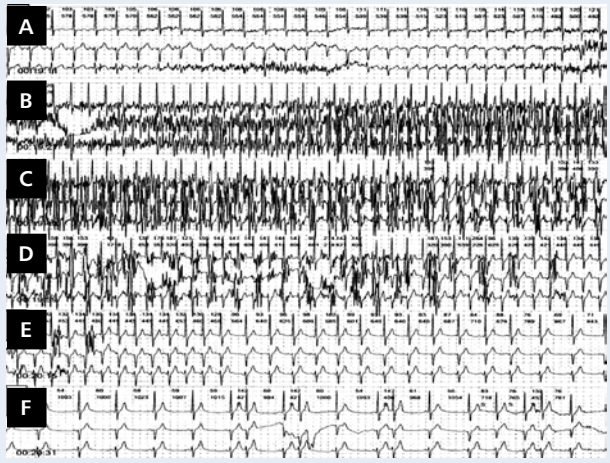

Figure 1. Continuous strips from Holter monitoring; **A**. Sinus tachycardia before seizures; **B**, **C**, **D**. Sinus tachycardia during seizures; **E**. Sinus rhythm with progressive heart rate reduction after the episode; **F**. Junctional rhythm with atrioventricular premature contractions

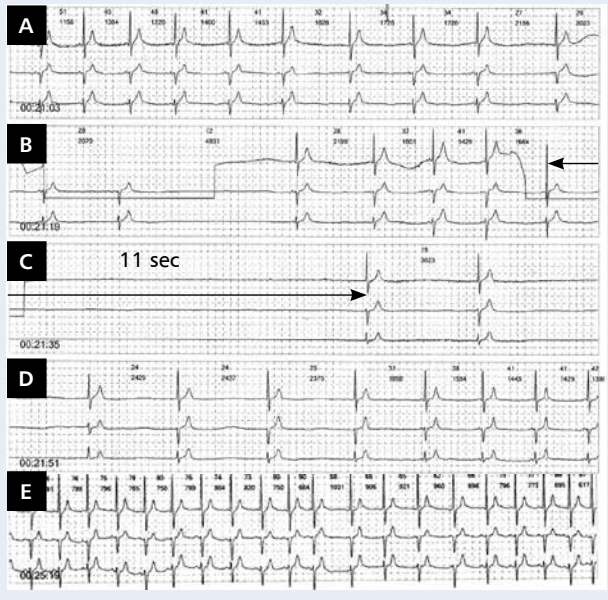

Figure 2. Continuous strips from Holter monitoring; **A**, **D**. Sinus bradycardia followed by a junctional escape rhythm; **B**, **C**. Pauses and asystole lasting 11 s; arrow shows episode of asystole; **E**. Sinus rhythm 3 min later, with heart rate between 60 bmp and 80 bpm

Address for correspondence:
Dr Adrian Baranchuk, MD, FACC, FRCPC, Associate Professor of Medicine and Physiology, Cardiac Electrophysiology and Pacing, Director, EP Training Program, Kingston General Hospital, Queen's University FAPC 3, 76 Stuart Street, K7L 2V7, Ontario, Canada, tel: 613 549 6666 ext. 3801, fax: 613 548 1387, e-mail: barancha@kgh.kari.net

Conflict of interest: *none declared*